close relationship between uric acid and the xanthin bases. The latter are simply antecedents of the former, containing less oxygen in the molecules and consequently being less highly oxidized products of metabolism. Horbaczewski was the first to show experimentally that uric acid was derived chiefly from the nuclein of the organism. He also demonstrated that if the oxidation of the nuclein be insufficient, xanthin bases and not uric acid resulted. Horbaczewski believed that the cell-nuclei in the body provided the nuclein from which the uric acid is derived, and further believed that uric acid was chiefly derived from leucocytes, which undergo destruction more readily than other cellular elements. He stated that there was a definite relationship between the leucocyte count and the amount of uric acid eliminated, an increase in the former being accompanied by a corresponding increase in the latter. The authors then refer to the work that has been done supporting or opposing Horbaczewski's view. Believing, themselves, that uric acid is derived chiefly from the nuclein resulting from the destruction of leucocytes, they held that diseased conditions associated with purulent exudations, in which this destruction is very marked, should show a marked increase in the excretion of uric acid. Such a condition is found in the resolution-stage of croupous pneumonia, where the products of leucocytic destruction are being constantly absorbed. Accordingly, they determined the uric acid elimination in five cases of pneumonia throughout the course of the disease. In all their cases the amount of uric acid began to increase the day before the crisis set in, and after the crisis suddenly increased so markedly that the amount eliminated was three times as great as during the febrile stage of the disease. This so-called "uric acid crisis" lasted from two to four days. For the next three or four days the amount eliminated gradually diminished, and by the seventh or eighth day after the crisis had once more reached normal. Their results accord with those of Ranke, Gerdes and Kühnan. Dunnin and Nowaczek did not count the leucocytes in their cases, so that it cannot be said that in these cases there is an association between the uric acid excretion and the number of leucocytes in the peripheral blood, as it is well known that the leucocytes usually fall to normal at the time or near the crisis. They believe, however, that their results strongly support Horbaczewski's view that the uric acid excretion is largely dependent on the nuclein derived from leucocyte destruction.

The Influence of Mitral Lesion on the Existence of Pulmonary Tuberculosis.—GRAHAM (*Montreal Medical Journal*, September, 1896) gives statistics compiled by a large number of observers showing the influence valvular lesions of the heart, particularly those of the mitral valve, have on the existence of pulmonary tuberculosis. There is much discrepancy in the results obtained from the statistics and in the opinions of many careful physicians.

In studying the clinical cases two difficulties present themselves: (1) the difficulty in diagnosis between mitral disease and pulmonary tuberculosis; (2) the trouble in deciding which is the primary lesion. French observers have shown that the pulmonary symptoms arising from mitral stenosis may closely simulate those of tuberculosis. Patients with the former may have dyspnœa, hemoptysis and emaciation, as well as local consolidation. The pulmonary engorgement may cause an elevation in temperature. Whether the heart-lesion is primary or secondary can only be decided by a very careful examination into the previous history as to the occurrence of rheumatism or not, or by the actual knowledge of the existence of the heart-lesion previous to the onset of the tuberculosis. The history of cases in which the two have coexisted during the middle period of life shows that the evolution of the tuberculosis was much delayed.

From the statistics and observations of eminent physicians the following conclusions may be arrived at:

(1) That mitral disease and pulmonary tuberculosis rarely exist in the same individual.

(2) That pulmonary tuberculosis is a very frequent sequel to pulmonary stenosis.

(3) The presence of mitral disease acts as a preventive to tuberculosis, especially when the vital powers are at or near their normal standard, and the prophylactic agency is shown in those who have a strong hereditary tendency to the disease, and whose surroundings are of a decidedly unhealthy character.

(4) In the small number of cases in which tuberculosis follows a mitral lesion during middle life its evolution is very much delayed. When, on the other hand, from age or bad habits, the vital powers are on the decline, the process is often as rapid as in ordinary cases.

There are a number of factors present in mitral lesions which may aid in the prophylaxis against tuberculosis:

(1) Increased pressure in the pulmonary circulation.

(2) The presence of transuded serum in the tissues, which renders foreign bodies more easily removed from the bronchial tubes, and which is believed by many to have a germicidal action.

(3) Increase in the involuntary muscular tissue, thus enabling the patient, more especially by coughing, to expel foreign matter from the alveoli and bronchi.

(4) The presence of an increased number of leucocytes in the alveoli.

Graham does not believe that the transuded serum has as marked a germicidal action as it is believed to possess. He states that in cirrhosis of the liver the same exudate exists in the peritoneal cavity, while it is well known that hepatic cirrhosis is frequently complicated by tuberculous peritonitis. He supports the view of Peters, that the passive congestion of the lungs acts as a prophylactic by producing an increased amount of chest measurement, especially at the apices, thus expanding the alveoli, under which circumstances there will be a diminished tendency to the development of tubercle.

The factors which Graham believes are of most importance in the prophylaxis against tubercle in mitral disease are:

(1) The increased amount of blood in the lungs.

(2) The greater expansion of the apices.

(3) The increase of the involuntary muscular fibres of the bronchial tubes and alveoli.

The reason why those who live in high altitudes are less liable to tuberculosis is attributed to the fact that it has been shown that in these persons there is a general hyperæmic condition of the lungs which is believed to retard the development of tuberculosis.